Shingles may increase risk of heart attack, stroke

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Occurrence of shingles may increase a person's composite risk of stroke and heart attack by 41%, according to a research letter.

Researchers in South Korea used the country's health services database to identify patients with newly diagnosed herpes zoster who experienced stroke or heart attack. The database covers the entire Korean population, and this subset consisted of approximately 570,000 people who received a medical check-up that provided comprehensive social and medical information. A total of 519,880 patients were followed from 2003 to 2013. There were 23,233 cases of shingles. A final cohort of 23,213 was matched with the same number of shingles-free patients to serve as control subjects. Results appeared in the July 11 *Journal of the American College of Cardiology*.

Patients with shingles were more likely to be female and have common risk factors for stroke and heart attack, such as old age, high blood pressure, diabetes and high cholesterol. But researchers also noted that this group was also less likely to smoke, had a lower alcohol intake, exercised more, and had higher socioeconomic status.

Shingles was found to be associated with an increased risk of cardiovascular events. The risk of a composite measure of events, including heart attack and stroke, was 41% higher. The risk of stroke specifically increased by 35%, and the risk of heart attack by 59%. The increase in risk for stroke was highest in those under 40 years old, a relatively younger population with fewer risks for atherosclerosis. The risks of both stroke and heart attack were highest the first year after the onset of shingles and decreased with time. However, these risks were evenly distributed in the shingles-free group.

Physicians treating patients with shingles should make them aware of the increased risk, the authors wrote. The authors cited several possible biological causes of stroke and myocardial infarction after herpes zoster, such as varicella-zoster virus replication adjacent to an artery, which leads to inflammation of the artery and subsequent thrombosis and rupture and repeated subclinical reactivation of varicella-zoster virus with a subsequent effect on the arteries.